

CHAPTER 38

Palpation of the Heart

KEY TEACHING POINTS

- A displaced supine apical impulse—defined as an impulse lateral to the midclavicular line (MCL)—is an accurate sign of an enlarged heart, reduced ejection fraction, and increased pulmonary capillary wedge pressure.
- Other measures of the displaced apical impulse (i.e., lateral to the nipple line or more than 10 cm from the midsternal line) are not as accurate as using the MCL for reference.
- In patients with mitral stenosis, the hyperkinetic apical movement indicates additional valvular lesions.
- In patients with chest pain or dyspnea, the sustained or double apical movement increases probability of left ventricular hypertrophy.
- Three different precordial movements increase the probability of moderate-to-severe tricuspid regurgitation: a lower sternal pulsation, a pulsatile liver, and the right ventricular rock.

I. INTRODUCTION

Much of the science of heart palpation is based on impulse cardiography and kinetocardiography, research tools from the 1960s that precisely timed normal and abnormal precordial movements and compared them with hemodynamic data and angiograms of the right ventricle and left ventricle (LVs). These precise and sensitive instruments could detect very small movements of the body wall, many of which are inconspicuous to the clinician's hand. Although this chapter refers to these studies to make certain points, only those movements easily palpable at the bedside are discussed.

Palpation of the heart is among the oldest physical examination techniques, having been recorded as early as 1550 BC by ancient Egyptian physicians (along with palpation of the peripheral pulses).¹ In the early 19th century Jean-Nicolas Corvisart, personal physician to Napoleon and teacher of Laennec, was the first to correlate cardiac palpation with postmortem findings and distinguish right ventricular enlargement from left ventricular enlargement.²⁻⁴ During animal experiments performed in 1830, James Hope proved that the cause of the apical impulse was ventricular contraction, which threw the heart up against the chest wall.⁵

II. TECHNIQUE

When palpating the chest, the clinician should describe the location, size, timing, and type of precordial movements.⁶

A. PATIENT POSITION

The clinician should first palpate the heart when the patient is lying supine and again with the patient lying on his or her left side. The supine position is used to locate all precordial movements and to identify whether these movements are abnormally hyperkinetic, sustained, or retracting (see later). The left lateral decubitus position is used to measure the diameter of the apical impulse and to detect additional abnormal diastolic filling movements (i.e., palpable third or fourth heart sounds).⁷

Because the left lateral decubitus position distorts the systolic apical movement, including those of healthy subjects (i.e., up to half of healthy patients have abnormally sustained movements in the lateral decubitus position), only the supine position should be used to characterize the patient's outward systolic movement.⁸

B. LOCATION OF ABNORMAL MOVEMENTS

Complete palpation of the heart includes four areas on the chest wall (Fig. 38.1).^{1,6,9-12}

1. **Apex Beat.** The apex beat or apical impulse is the palpable cardiac impulse farthest away from the sternum and farthest down on the chest wall, usually caused by the LV and located near the midclavicular line (MCL) in the fifth intercostal space. The clinician should also palpate the areas above and medial to the apex beat, where ventricular aneurysms sometimes become palpable.
2. **Left Lower Sternal Area (Fourth Intercostal Space Near Left Edge of Sternum).** Abnormal right ventricular and left atrial movements appear at this location.
3. **Left Base (Second Intercostal Space Near the Left Sternum).** Abnormal pulmonary artery movements or a palpable P_2 appear at this location.
4. **Right Base (Second Intercostal Space Near Right Edge of Sternum) and Sternoclavicular Joint.** Movements from an ascending aortic aneurysm may become palpable here.

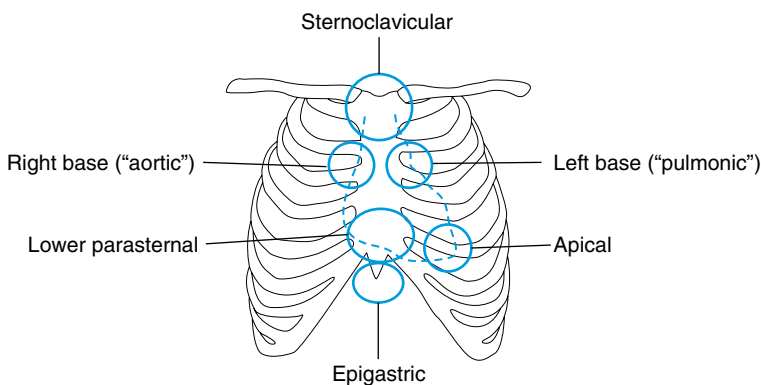


FIG. 38.1 LOCATIONS OF PRECORDIAL MOVEMENTS. The principal areas of precordial pulsations are the apical area, lower parasternal area, left base (i.e., second left intercostal parasternal space, “pulmonic area”), right base (i.e., second right intercostal parasternal space, “aortic area”), and sternoclavicular area. In some patients, especially those with chronic lung disease, right ventricular movements may appear in the epigastric area. The best external landmark is the sternal angle, which is where the second rib joins the sternum.

C. MAKING PRECORDIAL MOVEMENTS MORE CONSPICUOUS

Two teaching techniques are often used to bring out precordial movements and make them easier to time and characterize. In the first technique the clinician puts a dot of ink on the area of interest, whose direction and timing then become easy to see. In the second technique the clinician holds a cotton-tipped applicator stick against the chest wall, with the wooden end of the stick just off the center of the area of interest (the stick should be several inches long). The stick becomes a lever and the pulsating chest wall a fulcrum, causing the free end of the stick to trace in the air a magnified replica of the precordial movement. A folded paper stick-on note may substitute for the applicator stick.¹³

III. THE FINDINGS

Precordial movements are timed by simultaneously listening to the heart tones and noting the relationship between outward movements on the chest wall and the first and second heart sounds. There are four types of systolic movement: normal, hyperkinetic, sustained, and retracting.^{1,6,9-11}

A. NORMAL

The normal systolic movement is a small outward movement that begins with S_1 , ends by mid systole, and then retracts inward, returning to its original position long before S_2 .

The normal apical impulse is caused by a brisk early systolic anterior motion of the anterosseptal wall of the LV against the ribs.¹⁴ Despite its name, the apex beat bears no consistent relationship to the anatomic apex of the LV.¹⁴ In the supine position, the apex beat is palpable in only 25% to 40% of adults.¹⁵⁻¹⁸ In the lateral decubitus position, it is palpable in 50% to 73% of adults.^{15,19,20} The apex beat is more likely to be palpable in patients who have less body fat and who weigh less.²¹ Some studies show that the apical impulse is more likely to be present in women than men, but this difference disappears after controlling for the participants' weights.¹⁷

B. HYPERKINETIC

The hyperkinetic (or overacting) systolic movement is a movement identical in timing to the normal movement, although its amplitude is exaggerated. Distinguishing normal from hyperkinetic amplitude is a subjective process, even on precise tracings from impulse cardiography. This probably explains why the finding has minimal diagnostic value, appearing both in patients with volume overload of the LV (e.g., aortic regurgitation, ventricular septal defect) and in some normal persons who have thin chests or increased cardiac output.

C. SUSTAINED

The sustained movement is an abnormal outward movement that begins at S_1 but, unlike normal and hyperkinetic movements, extends to S_2 or even past it before beginning to descend to its original position. The amplitude of the sustained movement may be normal or increased. Sustained apical movements are always abnormal, indicating either pressure overload of the LV (e.g., aortic stenosis, severe hypertension), volume overload (e.g., aortic regurgitation, ventricular septal defect), a combination of pressure and volume overload (combined aortic stenosis and regurgitation), severe cardiomyopathy, or ventricular aneurysm.

D. RETRACTING

In the retracting movement, inward motion begins at S_1 and outward motion does not start until early diastole. Because retracting movements are sometimes identical to normal movements in every characteristic except for timing, they are easily overlooked unless the clinician listens to the heart tones when palpating the chest. Only two diagnoses cause the retracting impulse, constrictive pericarditis and severe tricuspid regurgitation.^{1,8,11}

E. HEAVES, LIFTS, AND THRUSTS

The words *heave* and *lift* sometimes refer to sustained movements and *thrust* to hyperkinetic ones, but these terms, often used imprecisely, are best avoided.^{1,9-11}

IV. CLINICAL SIGNIFICANCE

A. APEX BEAT

1. LOCATION

A traditional sign of an enlarged heart is an abnormally displaced apical impulse, which means it is located lateral to some external reference point. The three traditional reference points are: (1) the MCL, (2) a set distance from the midsternal line (the traditional upper limit of normal is 10 cm), and (3) the nipple line.

Of these three landmarks, the MCL is the best, as long as the clinician locates it precisely by palpating the acromioclavicular and sternoclavicular joints and marking the midpoint between them with a ruler.^{22,23} In the supine patient an apical impulse located outside the MCL increases the probability that the heart is enlarged on the chest radiograph (likelihood ratio [LR] = 3.4; [EBM Box 38.1](#)), the ejection fraction is reduced (LR = 10.3), the left ventricular end-diastolic volume is increased (LR = 5.1), and the pulmonary capillary wedge pressure is increased (LR = 5.8). Other studies confirm the relationship between displaced apical impulse and depressed ejection fraction.³¹

Using a point 10 cm from the midsternal line to define the displaced impulse is not a useful predictor of the enlarged heart (positive LR not significant, negative LR = 0.5; see [EBM Box 38.1](#)), probably because the 10 cm threshold is set too low (the MCL usually lies 10.5 to 11.5 cm from the midsternal line).²² Finally, the nipple line is the least reliable of the three landmarks, bearing no consistent relationship to the apical impulse or to the size of the chest, even in men. The distance of the nipple line from the midsternum or midclavicular line varies greatly.³²

2. DIAMETER OF THE APICAL IMPULSE

As measured in the left lateral decubitus position at 45 degrees, an apical impulse with a diameter of 4 cm or more increases the probability that the patient has a dilated heart (LR = 4.7 for increased left ventricular end-diastolic volume; see [EBM Box 38.1](#)). Smaller thresholds (e.g., 3 cm) discriminate between dilated and normal hearts in some studies, but not others.^{19,30}

3. ABNORMAL MOVEMENTS

A. HYPERKINETIC APICAL MOVEMENTS

The hyperkinetic apical movement is an important finding in one setting. In patients with mitral stenosis, left ventricular filling is impaired, causing the apical impulse to be normal or even reduced.³³ Therefore, if patients with the murmur of mitral stenosis also have a hyperkinetic apical impulse, an abnormality other than

**EBM BOX 38.1***Size and Position of Palpable Apical Impulse**

Finding (Reference) [†]	Sensitivity (%)	Specificity (%)	Likelihood Ratio [‡] if Finding Is	
			Present	Absent
Position of Apical Beat				
Supine Apical Impulse Lateral to MCL				
Detecting cardiothoracic ratio >0.5 ^{18,21,24}	39-60	76-93	3.4	0.6
Detecting low ejection fraction ²⁵⁻²⁸	5-66	93-99	10.3	0.7
Detecting increased left ventricular end-diastolic volume ^{20,29}	33-34	92-96	5.1	0.7
Detecting pulmonary capillary wedge pressure >12 mm Hg ²⁹	42	93	5.8	NS
Supine Apical Impulse >10 cm From Midsternal Line				
Detecting cardiothoracic ratio >0.5 ^{16,21,24}	61-80	28-97	NS	0.5
Size of Apical Beat				
Apical Beat Diameter ≥4 cm in Left Lateral Decubitus Position at 45 Degrees				
Detecting increased left ventricular end-diastolic volume ^{19,30}	48-85	79-96	4.7	NS

*Diagnostic standard: For *cardiothoracic ratio*, maximal transverse diameter of heart on chest radiography divided by maximal transverse diameter of thoracic cage; for *low ejection fraction*, LV ejection fraction <0.50²⁶ or <0.53²⁵ by scintigraphy, <0.5 by echocardiography,²⁸ or LV fractional shortening <25% by echocardiography;²⁷ for *increased LV end-diastolic volume*, >90 mL/m² or²⁹ >138 mL (echocardiography),³⁰ >109.2 mL/m² (computed tomography),²⁰ or upper fifth percentile of normal (echocardiography);¹⁹ for *increased LV mass*, LV mass by ultrafast computed tomography >191 g.¹⁵

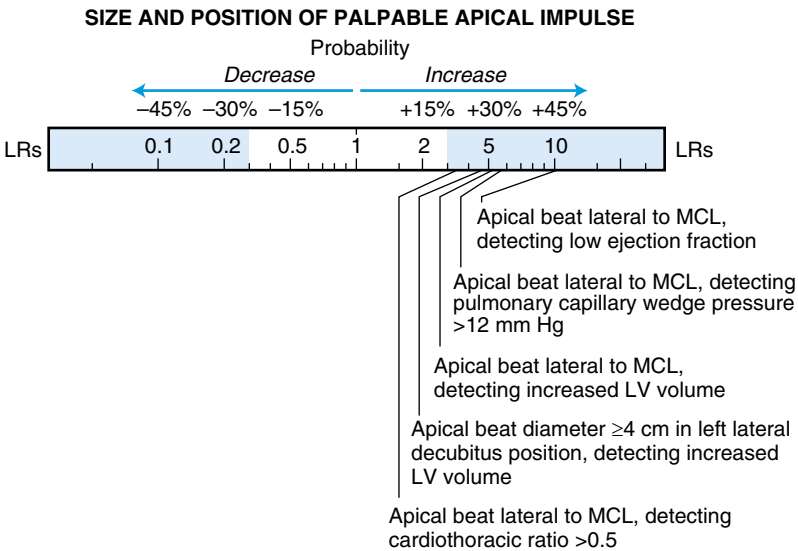
†Definition of findings: Except for "apical beat diameter," these data apply to all patients, whether or not an apical beat is palpable (i.e., nonpalpable apical beat = test "negative"). The only exception is the data for "apical beat diameter," which applies only to patients who have a measurable apical beat in the left lateral decubitus position (i.e., apical beat diameter ≥4 cm = test positive; <4 cm = test negative; unable to measure diameter = unable to evaluate using these data).

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

LV, Left ventricle; MCL, midclavicular line; NS, not significant.

[Click here to access calculator](#)

Continued



mitral stenosis also must be present, such as mitral regurgitation or aortic regurgitation (LR = 11.2; [EBM Box 38.2](#)).

B. SUSTAINED APICAL MOVEMENTS

A sustained or double apical movement (*double* refers to the combination of palpable S₄ and apical movement; see [Chapter 41](#)) increases the probability of left ventricular hypertrophy (LR = 5.6). In patients with aortic flow murmurs, the finding of a sustained apical impulse increases the probability of severe aortic stenosis (LR = 4.1; see [EBM Box 38.2](#)). In patients with the early diastolic murmur of aortic regurgitation, the sustained impulse is less helpful (LR = 2.4 for significant regurgitation), although the finding of a normal or absent apical impulse (i.e., not sustained or hyperkinetic) in these patients *decreases* significantly the probability of moderate-to-severe aortic regurgitation (LR = 0.1; see [EBM Box 38.2](#)).

C. RETRACTING APICAL IMPULSE

(1). CONSTRICTIVE PERICARDITIS. In up to 90% of patients with constrictive pericarditis, the apical impulse retracts during systole (sometimes accompanied by systolic retraction of the left parasternal area).^{8,40} In these patients the diseased pericardium prevents the normal outward systolic movement of the ventricles but allows rapid and prominent early diastolic filling of the ventricle. The prominent diastolic filling causes a palpable diastolic outward movement, which contributes to the overall impression that the apical impulse retracts during systole (see [Chapter 47](#)).

The first clinician to recognize the retracting apical impulse as a sign of “adhesive” pericarditis was Skoda in 1852.⁴¹

(2). TRICUSPID REGURGITATION. In severe tricuspid regurgitation a dilated right ventricle, occupying the apex, ejects blood into a dilated right atrium and liver, located nearer the sternum.⁸ This causes a characteristic rocking

**EBM BOX 38.2***Abnormal Palpable Movements**

Finding (Reference) [†]	Sensitivity (%)	Specificity (%)	Likelihood Ratio [‡] if Finding Is	
			Present	Absent
Hyperkinetic Apical Movement				
Detecting associated mitral regurgitation or aortic valve disease in patients with mitral stenosis ³³	74	93	11.2	0.3
Sustained or Double Apical Movement				
Detecting left ventricular hypertrophy ²⁰	57	90	5.6	0.5
Sustained Apical Movement				
Detecting severe aortic stenosis in patients with aortic flow murmurs ³⁴	78	81	4.1	0.3
Detecting moderate-to-severe aortic regurgitation in patients with basal early diastolic murmurs ³⁵	97	60	2.4	0.1
Lower Sternal Pulsations				
Detecting moderate to severe tricuspid regurgitation ³⁶	17	99	12.5	0.8
Sustained Left Lower Parasternal Movement				
Detecting right ventricular peak pressure ≥50 mm Hg ³⁷	71	80	3.6	0.4
Right Ventricular Rock				
Detecting moderate to severe tricuspid regurgitation ³⁶	5	100	31.4	NS
Pulsatile Liver				
Detecting moderate to severe tricuspid regurgitation ^{36,38}	12-30	92-99	6.5	NS
Palpable P₂				
Detecting pulmonary hypertension in patients with mitral stenosis ³⁹	96	73	3.6	0.05

Continued

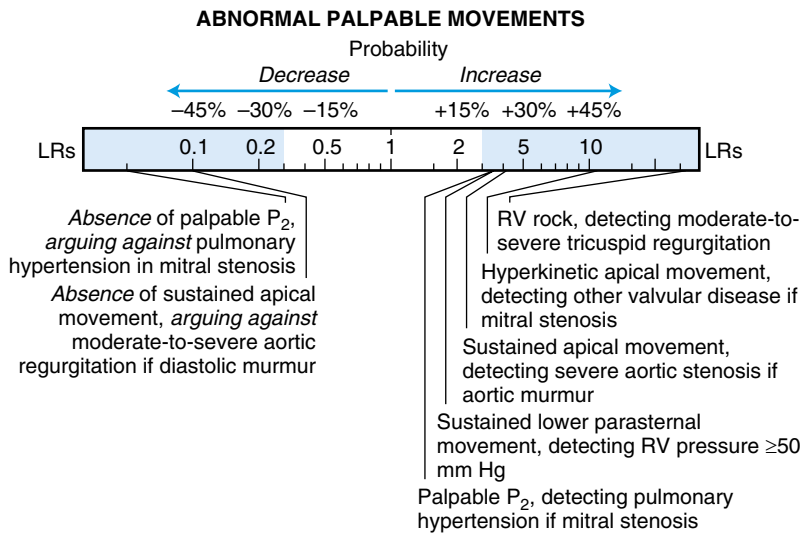
*Diagnostic standard: For *LV hypertrophy*, computed tomographic LV mass index > 104 g/m²,²⁰ for severe *aortic stenosis* and *moderate-to-severe aortic regurgitation*, see EBM Boxes in [Chapters 44 and 45](#); for *moderate-to-severe tricuspid regurgitation*, 3+ or 4+ by angiography³⁸ or as assessed visually from echocardiography,³⁶ and for *pulmonary hypertension*, mean pulmonary artery pressure ≥50 mm Hg.³⁹

[†]Definition of findings: For *abnormal apical movement*, “apical impulse heave or enlarged,”³⁵ “sustained,”³⁴ or “thrust”³³; for *sustained or double apical movement*, apical movement extending beyond S₂ or combination of palpable S₄ + LV apical movement;²⁰ for *abnormal parasternal movement*, “movement extending to or past S₂”;³⁷ for *right ventricular rock*, see the text; for *palpable P₂*, “palpable late systolic tap in second left intercostal space next to sternum, which frequently followed parasternal lift.”³⁹

[‡]Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

LV, Left ventricle; NS, not significant.

[Click here to access calculator](#)



motion (or right ventricular rock), the apical area retracting inward during systole and the lower left or right parasternal area moving outward during systole,⁴² often accompanied by a pulsatile liver. All three findings increase the probability of moderate-to-severe tricuspid regurgitation (LR = 31.4 for right ventricular rock, LR = 12.5 for lower sternal pulsations, and LR = 6.5 for pulsatile liver; see [EBM Box 38.2](#)).

B. LEFT LOWER PARASTERNAL MOVEMENTS

In normal persons the clinician either palpates no movement or only a tiny inward one during systole at this location. Abnormal movements at this location are classified as hyperkinetic or sustained, depending on their relationship to S₂.

I. HYPERKINETIC MOVEMENTS

Hyperkinetic movements of the left lower parasternal area occur in up to 50% of patients with atrial septal defect, which causes volume overload of the right

ventricle.⁴³ Nonetheless, this finding has limited diagnosis value without other findings of atrial septal defect—exaggerated y descent in the neck veins, wide and fixed S_2 splitting, and midsystolic murmur at the left second intercostal space (usually of grade 2 of 6)—because it is also sometimes found in patients without heart disease, such as those with thin chests, pectus excavatum, fever, or other high output states.^{37,43}

2. SUSTAINED MOVEMENTS

Sustained movements of the left lower sternal area may represent either an abnormal right ventricle (e.g., pressure overload from pulmonary hypertension or pulmonary stenosis or volume overload from atrial septal defect) or an enlarged left atrium (e.g., severe mitral regurgitation). Both right ventricular and left atrial parasternal movements are outward movements that begin to move inward only at S_2 or just after it and therefore are classified as sustained; they are distinguished by when the outward movement *begins*.

A. RIGHT VENTRICLE

Outward right ventricular movements begin at the first heart sound. If the clinician can exclude volume overload of the right ventricle and mitral regurgitation (both of which also cause parasternal movements), the finding of a sustained left parasternal movement is a modest sign of pulmonary hypertension (often accompanied by tricuspid regurgitation; see [Tricuspid Regurgitation](#), page 322). In patients with mitral stenosis, the duration of the sustained lower parasternal movement correlates well with pulmonary pressures.³³ In patients with a wide variety of valvular and congenital heart lesions (excluding mitral regurgitation), the sustained lower left parasternal movement is a modest discriminator between those with peak right ventricular pressures greater than 50 mm Hg and those with lower pressures (positive LR = 3.6, negative LR 0.4; see [EBM Box 38.2](#)). In patients with chronic liver disease undergoing evaluation for liver transplantation, the right ventricular heave increases the probability of pulmonary hypertension (i.e., mean pulmonary artery pressures of 25 mm Hg or more, LR = 8.8; see [Chapter 8](#)).⁴⁴ Up to 30% of patients with atrial septal defect, whether or not there is associated pulmonary hypertension, also have sustained lower left parasternal movements.⁴³

B. LEFT ATRIUM AND MITRAL REGURGITATION

In patients with severe mitral regurgitation, ventricular contraction forces blood backward into a dilated left atrium, which lies on the posterior surface of the heart and acts like an expanding cushion to lift up the heart, including the left parasternal area. This sustained movement, most easily palpated in the fourth or fifth intercostal space near the sternum,^{45,46} differs from those caused by the right ventricle, because outward movement begins in the second half of systole (it parallels the V wave on the left atrial pressure tracing).

In patients with isolated mitral regurgitation, the degree of the late systolic outward movement at the lower sternal edge correlates well with the severity of mitral regurgitation ($r = 0.93$, $p < 0.01$; the correlation is much worse if there is associated mitral stenosis, which may cause parasternal movements from pulmonary hypertension).^{45,46} In pure mitral regurgitation, as in atrial septal defect, the parasternal movement has no relationship to right ventricular pressures.⁴⁷

C. ANEURYSMS

In one study of consecutive patients with ventricular aneurysms identified by angiography, 33% had abnormal precordial movements.⁴⁸ Typical findings were: (1) a

double cardiac impulse, the first component representing the normal apical outward movement and the second the bulging of the aneurysm during peak ventricular pressures later in systole,^{49,50} and (2) a sustained impulse which extended superiorly or medially from the usual location of the apical impulse.⁴⁸ If detectable by palpation, the aneurysm originates in the anterior wall or apex of the LV; aneurysms originating from the inferior or lateral wall are too distant from the anterior chest wall to be detectable by palpation.⁴⁸

D. DIFFUSE PRECORDIAL MOVEMENTS

Diffuse outward movements of the entire precordium, from the apex to lower parasternal area, may result from (1) right ventricular enlargement (which dilates to occupy the apical area), (2) left ventricular enlargement (which rotates to occupy the lower parasternal area) or (3) biventricular enlargement.¹¹ Palpation alone cannot distinguish these different etiologies—even sensitive recordings from impulse cardiography or kinetocardiography could not do this—and the clinician must rely on other findings to determine which chamber is most likely causing the diffuse movement.

E. RIGHT LOWER PARASTERNAL MOVEMENTS

Abnormal systolic outward movements appear in the right lower parasternal area from tricuspid regurgitation (ejection of blood into the right atrium and liver, which lies under the right side of the sternum) or from mitral regurgitation (ejection of blood in a dilated left atrium).^{11,42,51}

F. PALPABLE P₂

A palpable P₂ (i.e., the pulmonic component of second heart sound) is a sharp, brief snapping sensation felt over the left base, coincident with S₂. It is much briefer than other precordial movements. In patients with mitral stenosis a palpable P₂ increases the probability of pulmonary hypertension (LR = 3.6 for mean pulmonary pressure >50 mm Hg). More importantly, the absence of a palpable P₂ in these patients decreases the probability of a pulmonary pressure this high (LR = 0.05; see [EBM Box 38.2](#)).

G. PALPABLE THIRD AND FOURTH HEART SOUNDS

Some patients with rapid early ventricular filling (e.g., mitral regurgitation) have a palpable early diastolic movement at the apex. Other patients with strong atrial contractions into stiff ventricles (e.g., hypertensive or ischemic heart disease) have palpable presystolic apical movements. These movements have the same significance as their audible counterparts, the third and fourth heart sound (i.e., S₃ and S₄; see [Chapter 41](#)). They are usually called *palpable S₃* and *palpable S₄*.

The S₄ is much more likely to be palpable than the S₃, and both are more likely to be felt when the patient is in the lateral decubitus position.^{7,9,10} The palpable S₄ causes either a double outward impulse near S₁ (a common analogy is the grace note in music; see *double apical movement* in [EBM Box 38.2](#)) or single outward movement, consisting of the palpable S₄ and apical beat together, which is distinguished from the apical beat alone because the outward movement begins slightly before S₁.^{10,11}

The references for this chapter can be found on www.expertconsult.com.

REFERENCES

1. Basta LL, Bettinger JJ. The cardiac impulse: a new look at an old art. *Am Heart J*. 1979;97(1):96–111.
2. Stokes W. *An Introduction to the Use of the Stethoscope*. (facsimile edition by the Classics of Cardiology Library). Edinburgh: Maclachlin and Stewart; 1825.
3. Corvisart JN. *An Essay on the Organic Diseases and Lesions of the Heart and Great Vessels*. (facsimile edition by New York Academy of Medicine). Boston, MA: Bradford and Read; 1812.
4. Willius FA, Dry TJ. *A History of the Heart and the Circulation*. Philadelphia, PA: W. B. Saunders Co.; 1948.
5. McCrady JD, Hoff HE, Geddes LA. The contributions of the horse to knowledge of the heart and circulation: IV. James Hope and the heart sounds. *Conn Med*. 1966;30(2):126–131.
6. Feinstein AR, Hochstein E, Luisada AA, et al. Glossary of cardiologic terms related to physical diagnosis and history. Part III—anterior chest movements. *Chest*. 1969;56(3):231–232.
7. Bethell HJN, Nixon PGF. Examination of the heart in supine and left lateral positions. *Br Heart J*. 1973;35:902–907.
8. Boicourt OW, Nagle RE, Mounsey JPD. The clinical significance of systolic retraction of the apical impulse. *Br Heart J*. 1965;27:379–391.
9. Mounsey JPD. Inspection and palpation of the cardiac impulse. *Prog Cardiovasc Dis*. 1967;10(3):187–206.
10. Mounsey P. Praecordial pulsations in health and disease. *Postgrad Med J*. 1968;44:134–139.
11. Stapleton JF, Groves BM. Precordial palpation. *Am Heart J*. 1971;81(3):409–427.
12. Willis PW. Analysis of precordial movements. *Heart Dis Stroke*. 1993;2:284–289.
13. Shindler D. Post-it apexcardiography. *N Engl J Med*. 2004;351:1364.
14. Deliyannis AA, Gillam PMS, Mounsey JPD, Steiner RE. The cardiac impulse and the motion of the heart. *Br Heart J*. 1964;26:396–411.
15. Heckerling PS, Wiener SL, Wolfkiel CJ, et al. Accuracy and reproducibility of precordial percussion and palpation for detecting increased left ventricular end-diastolic volume and mass. A comparison of physical findings and ultrafast computed tomography of the heart. *J Am Med Assoc*. 1993;270(16):1943–1948.
16. Heckerling PS, Wiener SL, Moses VK, Claudio J, Kushner MS, Hand R. Accuracy of precordial percussion in detecting cardiomegaly. *Am J Med*. 1991;91:328–334.
17. Niehaus FW, Wright WD. Facts and fallacies about the normal apex beat. *Am Heart J*. 1945;30:604–609.
18. Mulkerrin E, Saran R, Dewar R, Harding JR, Bayer AJ, Finucane P. The apex cardiac beat: not a reliable clinical sign in elderly patients. *Age Ageing*. 1991;20(4):304–306.
19. Dans AL, Bossone EF, Guyatt GH, Fallen EL. Evaluation of the reproducibility and accuracy of apex beat measurement in the detection of echocardiographic left ventricular dilation. *Can J Cardiol*. 1995;11(6):493–497.
20. Ehara S, Okuyama T, Shirai N, et al. Comprehensive evaluation of the apex beat using 64-slice computed tomography: impact of left ventricular mass and distance to chest wall. *J Cardiol*. 2010;55:256–265.
21. O'Neill TW, Smith M, Barry M, Graham IM. Diagnostic value of the apex beat. *Lancet*. 1989;1(8635):410–411.
22. Naylor CD, McCormack DG, Sullivan SN. The midclavicular line: a wandering landmark. *Can Med Assoc J*. 1987;136:48–50.
23. Ryand DA. The midclavicular line: where is it? *Ann Intern Med*. 1968;69:329–330.
24. O'Neill TW, Barry MA, Smith M, Graham IM. Diagnostic value of the apex beat. *Lancet*. 1989;2(8661):499.
25. Gadsboll N, Hoiland-Carlsen PF, Nielsen GG, et al. Interobserver agreement and accuracy of bedside estimation of right and left ventricular ejection fraction in acute myocardial infarction. *Am J Cardiol*. 1989;63:1301–1307.
26. Mattleman SJ, Hakki AH, Iskandrian AS, Segal BL, Kane SA. Reliability of bedside evaluation in determining left ventricular function: correlation with left ventricular ejection fraction determined by radionuclide ventriculography. *J Am Coll Cardiol*. 1983;1(2):417–420.

27. Davie AP, Caruana FL, Sutherland GR, McMurray JJV. Assessing diagnosis in heart failure: which features are any use? *Q J Med.* 1997;90:335–339.
28. Fahey T, Jeyaseelan S, McCowan C, et al. Diagnosis of left ventricular systolic dysfunction (LVSD): development and validation of a clinical prediction rule in primary care. *Fam Pract.* 2007;24:628–635.
29. Gadsboll N, Hoilund-Carsen PF, Nielsen GG, et al. Symptoms and signs of heart failure in patients with myocardial infarction: reproducibility and relationship to chest X-ray, radionuclide ventriculography and right heart catheterization. *Eur Heart J.* 1989;10:1017–1028.
30. Eilen SD, Crawford MH, O'Rourke RA. Accuracy of precordial palpation for detecting increased left ventricular volume. *Ann Intern Med.* 1983;99:628–630.
31. Eagle KA, Quettermous T, Singer DE, et al. Left ventricular ejection fraction. Physician estimates compared with gated blood pool scan measurements. *Arch Intern Med.* 1988;148:882–885.
32. Kurtz CM, White PD. The percussion of the heart borders and the Roentgen ray shadow of the heart. *Am J Med Sci.* 1928;176:181–195.
33. Wood P. An appreciation of mitral stenosis: part 1. Clinical features. Part 2. Investigations and results. *Br Med J.* 1954;1:1051–1063, 1113–1124.
34. Forssell G, Jonasson R, Orinius E. Identifying severe aortic valvular stenosis by bedside examination. *Acta Med Scand.* 1985;218:397–400.
35. Frank MJ, Casanegra P, Migliori AJ, Levinson GE. The clinical evaluation of aortic regurgitation. *Arch Intern Med.* 1965;116:357–365.
36. McGee SR. Etiology and diagnosis of systolic murmurs in adults. *Am J Med.* 2010;123:913–921.
37. Gillam PMS, Deliyannis AA, Mounsey JPD. The left parasternal impulse. *Br Heart J.* 1964;26:726–736.
38. Cha SD, Gooch AS. Diagnosis of tricuspid regurgitation. *Arch Intern Med.* 1983;143:1763–1768.
39. Whitaker W. Clinical diagnosis of pulmonary hypertension in patients with mitral stenosis. *Q J Med.* 1954;23:105–112.
40. El-Sherif A, El-Said G. Jugular, hepatic, and praecordial pulsations in constrictive pericarditis. *Br Heart J.* 1971;33:305–312.
41. Skoda J. *Auscultation and Percussion*. Philadelphia, PA: Lindsay and Blakiston; 1854.
42. Salazar E, Levine HD. Rheumatic tricuspid regurgitation: the clinical spectrum. *Am J Med.* 1962;33:111–129.
43. Fukumoto T, Ito M, Arita M, Tetsuo M, Fujino T, Mashiba H. Right parasternal lift in atrial septal defect. *Am Heart J.* 1977;94(6):699–704.
44. Pilatis ND, Jacobs LE, Rekpattanapipat P, et al. Clinical predictors of pulmonary hypertension in patients undergoing liver transplant evaluation. *Liver Transpl.* 2000;6:85–91.
45. James TM, Swartzell RH, Eddleman EE. Hemodynamic significance of the precordial late systolic outward movement in mitral regurgitation. *Ala J Med Sci.* 1978;15(1):55–64.
46. Basta LL, Wolfson P, Eckberg DL, Abboud FM. The value of left parasternal impulse recordings in the assessment of mitral regurgitation. *Circulation.* 1973;48:1055–1065.
47. Manchester GH, Block P, Gorlin R. Misleading signs in mitral insufficiency. *J Am Med Assoc.* 1965;191(2):99–100.
48. Gorlin R, Klein MD, Sullivan JM. Prospective correlative study of ventricular aneurysm: mechanistic concept and clinical recognition. *Am J Med.* 1967;42:512–531.
49. El-Sherif A, Saad Y, El-Said G. Praecordial tracings of myocardial aneurysms. *Br Heart J.* 1969;31:357–364.
50. Eddleman EE, Langley JO. Paradoxical pulsation of the precordium in myocardial infarction and angina pectoris. *Am Heart J.* 1962;63(5):579–581.
51. El-Sherif N, El-Ramly Z. External left atrial pulse tracings in extreme left atrial dilation. *Am Heart J.* 1972;84(3):387–394.